PALM INTRANET

Day : Friday
Date: 1/30/2004

Time: 09:51:01

# Inventor Name Search

Enter the **first few letters** of the Inventor's Last Name.
Additionally, enter the **first few letters** of the Inventor's First name.

| **Last Name** | **First Name** | |
|---|---|---|
| Croze | Ed | Search |

To go back use Back button on your browser toolbar.

Back to PALM | ASSIGNMENT | OASIS | Home page

h e eb cg b e f e

# Freeform Search

**Database:** US Pre-Grant Publication Full-Text Database; US Patents Full-Text Database; US OCR Full-Text Database; EPO Abstracts Database; JPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

**Term:** L5 and (potentiating or enhancing or increasing)

**Display:** 10 **Documents in Display Format:** - **Starting with Number** 1

**Generate:** ○ Hit List ◉ Hit Count ○ Side by Side ○ Image

Search Clear Interrupt

## Search History

**DATE: Friday, January 30, 2004** Printable Copy Create Case

| Set Name side by side | Query | Hit Count | Set Name result set |
|---|---|---|---|
| *DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; THES=ASSIGNEE; PLUR=YES; OP=AND* | | | |
| L6 | L5 and (potentiating or enhancing or increasing) | 186 | L6 |
| L5 | L4 and (cancer or tumor or tumour) | 195 | L5 |
| L4 | (Interferon adj receptor) same (vector or (nucleic adj acid)) | 202 | L4 |
| L3 | (IFNAR2c) | 4 | L3 |
| L2 | (interferon adj receptor) adj (2 adj chain) | 0 | L2 |
| L1 | Croze-Ed.in. | 1 | L1 |

END OF SEARCH HISTORY

### Status: Path 1 of [Dialog Information Services via Modem]

### Status: Initializing TCP/IP using (UseTelnetProto 1 ServiceID pto-dialog)
Trying 31060000009999...Open

DIALOG INFORMATION SERVICES
PLEASE LOGON:
******** HHHHHHHH SSSSSSSS?
### Status: Signing onto Dialog
********
ENTER PASSWORD:
******** HHHHHHHH SSSSSSSS? ********
Welcome to DIALOG
### Status: Connected

Dialog level 03.07.00D

Last logoff: 23jan04 16:44:47
Logon file001 30jan04 11:26:00
*** ANNOUNCEMENT ***
***
--File 654 - US published applications from March 15, 2001 to the present are now online. Please see HELP NEWS 654 for details.

***
--File 581 - The 2003 annual reload of Population Demographics is complete. Please see Help News581 for details.

***
--File 990 - NewsRoom now contains February 2003 to current records. File 992 - NewsRoom 2003 archive has been newly created and contains records from January 2003. The oldest months's records roll out of File 990 and into File 992 on the first weekend of each month. To search all 2003 records BEGIN 990, 992, or B NEWS2003, a new OneSearch category.
***
--Connect Time joins DialUnits as pricing options on Dialog. See HELP CONNECT for information.
***
***
--SourceOne patents are now delivered to your email inbox as PDF replacing TIFF delivery. See HELP SOURCE1 for more information.
***
--Important news for public and academic libraries. See HELP LIBRARY for more information.
***
--Important Notice to Freelance Authors--
See HELP FREELANCE for more information
***
NEW FILES RELEASED
***DIOGENES: Adverse Drug Events Database (File 181)
***World News Connection (File 985)
***Dialog NewsRoom - 2003 Archive (File 992)
***TRADEMARKSCAN-Czech Republic (File 680)
***TRADEMARKSCAN-Hungary (File 681)
***TRADEMARKSCAN-Poland (File 682)
***
UPDATING RESUMED
***
RELOADED
***Population Demographics -(File 581)
***CLAIMS Citation (Files 220-222)

```
REMOVED
                    ***

      >>> Enter BEGIN HOMEBASE for Dialog Announcements <<<
      >>>    of new databases, price changes, etc.       <<<
                    ****
KWIC is set to 50.
HILIGHT set on as '*'
* * * ALL NEW CURRENT YEAR RANGES HAVE BEEN * * *
* * * INSTALLED   * * *

File   1:ERIC  1966-2004/Jan 20
       (c) format only 2004 The Dialog Corporation

      Set  Items  Description
      ---  -----  -----------
Cost is in DialUnits
?b 155, 159, 5, 73
       30jan04 11:26:13 User259876 Session D587.1
            $0.32    0.090 DialUnits File1
     $0.32  Estimated cost File1
     $0.04  TELNET
     $0.36  Estimated cost this search
     $0.36  Estimated total session cost   0.090 DialUnits


SYSTEM:OS  - DIALOG OneSearch
  File 155:MEDLINE(R)  1966-2004/Jan W4
         (c) format only 2004 The Dialog Corp.
*File 155: Medline is updating again (12-22-2003).
Please see HELP NEWS 154, for details.
  File 159:Cancerlit  1975-2002/Oct
         (c) format only 2002 Dialog Corporation
*File 159: Cancerlit ceases updating with immediate effect.
Please see HELP NEWS.
  File   5:Biosis Previews(R)  1969-2004/Jan W4
         (c) 2004 BIOSIS
  File  73:EMBASE  1974-2004/Jan W4
         (c) 2004 Elsevier Science B.V.

      Set  Items  Description
      ---  -----  -----------
?s (interferon (w) (therapy or treatment))
          344736  INTERFERON
         5318351  THERAPY
         4702358  TREATMENT
      S1   15712  (INTERFERON (W) (THERAPY OR TREATMENT))
?s s1 and (IFNAR2c)
           15712  S1
              39  IFNAR2C
      S2       0  S1 AND (IFNAR2C)
?s s1 and review
           15712  S1
         1675550  REVIEW
      S3     665  S1 AND REVIEW
?s s3 and (cancer and restenosis)
             665  S3
         2359825  CANCER
           29824  RESTENOSIS
      S4       0  S3 AND (CANCER AND RESTENOSIS)
?s s3 and (restenosis)
             665  S3
           29824  RESTENOSIS
      S5       0  S3 AND (RESTENOSIS)
?s s3 and (cancer)
             665  S3
         2359825  CANCER
```

```
      S6      140  S3 AND (CANCER)
?s s6 and (gene (w) therapy)
Processing
             140  S6
         2403862  GENE
         5318351  THERAPY
           78696  GENE(W)THERAPY
      S7       1  S6 AND (GENE (W) THERAPY)
?t s7/3,k/all
```

**7/3,K/1 (Item 1 from file: 73)**

```
DIALOG(R)File  73:EMBASE
(c) 2004 Elsevier Science B.V. All rts. reserv.

07200773      EMBASE No: 1998100713
```

**Bone marrow transplantation for chronic myelogenous leukemia**

```
  Enright H.; McGlave P.
  Dr. H. Enright, The Adelaide Hospital, Dublin 24  Ireland
  Current Opinion in Oncology ( CURR. OPIN. ONCOL. ) (United States)  1998
, 10/2 (100-107)
  CODEN: CUOOE   ISSN: 1040-8746
  DOCUMENT TYPE: Journal; Review
  LANGUAGE: ENGLISH   SUMMARY LANGUAGE: ENGLISH
  NUMBER OF REFERENCES: 75

  ...malignancy characterized by an initial chronic phase of expanded
clonal myelopoiesis followed by inevitable progression to accelerated phase
and finally to fatal blast crisis. Although *interferon* *therapy* results
in hematologic control of disease in most patients and major cytogenetic
responses in 30% to 35%, resulting in better survival than with
conventional therapy...
MEDICAL DESCRIPTORS:
minimal residual disease; peripheral blood stem cell; autotransplantation;
*cancer* recurrence; graft versus leukemia effect; *gene* *therapy*;
immunomodulation; human; *review*; priority journal
SECTION HEADINGS:
  016  *Cancer*
  025  Hematology
  037  Drug Literature Index
?ds

Set      Items   Description
S1       15712   (INTERFERON (W) (THERAPY OR TREATMENT))
S2           0   S1 AND (IFNAR2C)
S3         665   S1 AND REVIEW
S4           0   S3 AND (CANCER AND RESTENOSIS)
S5           0   S3 AND (RESTENOSIS)
S6         140   S3 AND (CANCER)
S7           1   S6 AND (GENE (W) THERAPY)
?s (IFNAR2c)
      S8      39  (IFNAR2C)
?s s8 and (vector)
              39  S8
          276766  VECTOR
      S9       1  S8 AND (VECTOR)
?t s9/3,k/all
```

**9/3,K/1 (Item 1 from file: 5)**

```
DIALOG(R)File   5:Biosis Previews(R)
(c) 2004 BIOSIS. All rts. reserv.

0013657475   BIOSIS NO.: 200200250986
```

**Interferon-alpha activates multiple STAT signals and down-regulates c-Met in primary human hepatocytes**

```
AUTHOR: Radaeva Svetlana; Jaruga Barbara; Hong Feng; Kim Won-Ho; Fan Saijun
  ; Cai Hongbo; Strom Stephen; Liu Youhua; El-Assal Osama; Gao Bin
  (Reprint)
```

AUTHOR ADDRESS: Section on Liver Biology, NIAAA, NIH, 12420 Parklawn Drive, Park Building Room 120, MSC 8115, Bethesda, MD, 20892, USA**USA
JOURNAL: Gastroenterology 122 (4): p1020-1034 April, 2002 2002
MEDIUM: print
ISSN: 0016-5085
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

...ABSTRACT: The differential response to IFN-alpha stimulation in primary human and mouse hepatocytes may be caused by expression of predominant functional IFN-alpha receptor 2c (*IFNAR2c*) in primary human hepatocytes vs. expression of predominant inhibitory IFNAR2a in mouse hepatocytes. Microarray analyses of primary human hepatocytes show that IFN-alpha up-regulates...
DESCRIPTORS:
CHEMICALS & BIOCHEMICALS: ...interferon-alpha receptor 2c {*IFNAR2c*};
...METHODS & EQUIPMENT: expression/*vector* techniques, gene transfer method
?ds

| Set | Items | Description |
|---|---|---|
| S1 | 15712 | (INTERFERON (W) (THERAPY OR TREATMENT)) |
| S2 | 0 | S1 AND (IFNAR2C) |
| S3 | 665 | S1 AND REVIEW |
| S4 | 0 | S3 AND (CANCER AND RESTENOSIS) |
| S5 | 0 | S3 AND (RESTENOSIS) |
| S6 | 140 | S3 AND (CANCER) |
| S7 | 1 | S6 AND (GENE (W) THERAPY) |
| S8 | 39 | (IFNAR2C) |
| S9 | 1 | S8 AND (VECTOR) |

?s s8 and (cancer or tumor or tumour)

| | | |
|---|---|---|
| | 39 | S8 |
| | 2359825 | CANCER |
| | 2373338 | TUMOR |
| | 281873 | TUMOUR |
| S10 | 18 | S8 AND (CANCER OR TUMOR OR TUMOUR) |

?rd
...completed examining records

| | | |
|---|---|---|
| S11 | 9 | RD (unique items) |

?t s11/3,k/all

**11/3,K/1 (Item 1 from file: 155)**
DIALOG(R)File 155:MEDLINE(R)


11724200 99160899 PMID: 10049744
**Formation of a uniquely stable type I interferon receptor complex by interferon beta is dependent upon particular interactions between interferon beta and its receptor and independent of tyrosine phosphorylation.**
Russell-Harde D; Wagner T C; Perez H D; Croze E
Department of Protein Biochemistry, Department of Immunology, Berlex Biosciences, Richmond, California 94804, USA.
Biochemical and biophysical research communications (UNITED STATES) Feb 16 1999, 255 (2) p539-44, ISSN 0006-291X Journal Code: 0372516
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Human type I interferons (IFN) require two receptor chains, IFNAR1 and *IFNAR2c* for high affinity (pM) binding and biological activity. Our previous studies have shown that the ligand dependent assembly of the type I IFN receptor chains...

...for all type I IFNs. IFNbeta appears unique in its ability to assemble a

stable complex of receptor chains, as demonstrated by the observation that *IFNAR2c* co-immunoprecipitates with IFNAR1 when cells are stimulated with IFNbeta but not with IFNalpha. The characteristics of such a receptor complex are not well defined...

... receptor assembly. To further characterize the factors required for formation of such a stable receptor complex we demonstrate using IFN stimulated Daudi cells that (1) *IFNAR2c* co-immunoprecipitates with IFNAR1 even when tyrosine phosphorylation of receptor chains is blocked with staurosporine, and (2) IFNbeta1b but not IFNalpha2, is present in the...
; Interferon Type I, Recombinant--metabolism--ME; Macromolecular Systems; Models, Biological; Models, Molecular; Phosphorylation; *Tumor* Cells, Cultured

**11/3,K/2 (Item 2 from file: 155)**
DIALOG(R)File 155:MEDLINE(R)


10188030 22218008 PMID: 12105218
**STAT3 activation by type I interferons is dependent on specific tyrosines located in the cytoplasmic domain of interferon receptor chain 2c. Activation of multiple STATS proceeds through the redundant usage of two tyrosine residues.**
Velichko Sharlene; Wagner T Charis; Turkson James; Jove Richard; Croze Ed
Department of Immunology, Berlex Biosciences Inc., Richmond, California 94804 and the Molecular Oncology and Drug Discovery Programs, H. Lee Moffitt Cancer Center and Research Institute, Tampa, Florida 33612.
Journal of biological chemistry (United States) Sep 20 2002, 277 (38) p35635-41, ISSN 0021-9258 Journal Code: 2985121R
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... remains unclear. Understanding the IFN-dependent regulation of STAT3 is of increasing interest because recent studies have demonstrated that STAT3 may play a role in *cancer*. Studies have revealed that STAT3 is constitutively active in a number of *cancer* cell lines and that overexpression of an active form of STAT3 transforms normal fibroblasts. Therefore, STAT3 exhibits properties indicative of known oncogenes. In this report...

...the role of the type I IFN receptor in STAT3 activation and identify for the first time tyrosine residues present in the cytoplasmic domain of *IFNAR2c* that are critical for STAT3 activation. The regulation of STAT3 activation by IFNs was measured in a human lung fibrosarcoma cell line lacking *IFNAR2c* but stably expressing various *IFNAR2c* tyrosine mutants. We show here that in addition to IFN-dependent tyrosine phosphorylation of STAT3, activation using a STAT3-dependent electrophoretic mobility shift assay and...

... type I IFN-dependent activation of STAT3 proceeds through a novel mechanism that is dependent on two tyrosines, Tyr(337) and Tyr(512), present in *IFNAR2c* and contained within a conserved six-amino acid residue motif, GxGYxM. Surprisingly, both tyrosines were previously shown to be required for type I IFN-dependent...

... activation. Our results reveal that type I IFNs activate multiple STATs via the overlapping usage of two tyrosine residues located in the cytoplasmic domain of *IFNAR2c*.
; Base Sequence; DNA Primers; Electrophoretic Mobility Shift Assay; Receptors, Interferon--chemistry--CH; *Tumor* Cells, Cultured

**11/3,K/3 (Item 3 from file: 155)**
DIALOG(R)File 155:MEDLINE(R)

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09980887 21906790 PMID: 11910354

**Interferon-alpha activates multiple STAT signals and down-regulates c-Met in primary human hepatocytes.**

Radaeva Svetlana; Jaruga Barbara; Hong Feng; Kim Won-Ho; Fan Saijun; Cai Hongbo; Strom Stephen; Liu Youhua; El-Assal Osama; Gao Bin

Section on Liver Biology, Laboratory of Physiologic Studies, National Institute on Alcohol Abuse and Alcoholism, National Institutes of Health, Bethesda, Maryland 20892, USA.

Gastroenterology (United States) Apr 2002, 122 (4) p1020-34, ISSN 0016-5085 Journal Code: 0374630

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

... The differential response to IFN-alpha stimulation in primary human and mouse hepatocytes may be caused by expression of predominant functional IFN-alpha receptor 2c (*IFNAR2c* ) in primary human hepatocytes vs. expression of predominant inhibitory IFNAR2a in mouse hepatocytes. Microarray analyses of primary human hepatocytes show that IFN-alpha up-regulates about 44 genes by over 2-fold and down-regulates about 9 genes by 50%. The up-regulated genes include a variety of antiviral and *tumor* suppressors/proapoptotic genes. The down-regulated genes include c-myc and c-Met, the hepatocyte growth factor (HGF) receptor. Down-regulation of c-Met is...

...; Oligonucleotide Array Sequence Analysis; Proto-Oncogene Protein c-met--metabolism--ME; Rats; Rats, Sprague-Dawley; Receptors, Interferon --genetics--GE; Solubility; Transcription Factor, Sp1--metabolism--ME; *Tumor* Cells, Cultured; Up-Regulation--drug effects--DE

**11/3,K/4 (Item 4 from file: 155)**

DIALOG(R)File 155:MEDLINE(R)

(c) format only 2004 The Dialog Corp. All rts. reserv.

09193984 20501119 PMID: 11046044

**Receptor for activated C-kinase (RACK-1), a WD motif-containing protein, specifically associates with the human type I IFN receptor.**

Croze E; Usacheva A; Asarnow D; Minshall R D; Perez H D; Colamonici O

Department of Immunology, Berlex Biosciences, Richmond CA 94804, USA. ed croze@berlex.com

Journal of immunology (Baltimore, Md. - 1950) (UNITED STATES) Nov 1 2000, 165 (9) p5127-32, ISSN 0022-1767 Journal Code: 2985117R

Contract/Grant No.: CA55079; CA; NCI; GM54709; GM; NIGMS

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

The cytoplasmic domain of the human type I IFN receptor chain 2 ( *IFNAR2c* or IFN-alphaRbetaL) was used as bait in a yeast two-hybrid system to identify novel proteins interacting with this region of the receptor. We ...

...; isolation and purification--IP; Repetitive Sequences, Amino Acid --genetics--GE; Repetitive Sequences, Amino Acid--immunology--IM; Saccharomyces cerevisiae--genetics--GE; Tetradecanoylphorbol Acetate --pharmacology--PD; Tryptophan; *Tumor* Cells, Cultured; Two-Hybrid System Techniques

**11/3,K/5 (Item 5 from file: 155)**

DIALOG(R)File 155:MEDLINE(R)

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09081961 20379040 PMID: 10825167

**Role of the intracellular domain of the human type I interferon receptor 2 chain (*IFNAR2c*) in interferon signaling. Expression of *IFNAR2c* truncation mutants in U5A cells.**

Russell-Harde D; Wagner T C; Rani M R; Vogel D; Colamonici O; Ransohoff R M; Majchrzak B; Fish E; Perez H D; Croze E

Berlex Biosciences, Richmond, California 94804, the Cleveland Clinic Foundation, Cleveland, Ohio, 44195, USA.

Journal of biological chemistry (UNITED STATES) Aug 4 2000, 275 (31) p23981-5, ISSN 0021-9258 Journal Code: 2985121R

Contract/Grant No.: 2PO1 62220; PHS; CA55079; CA; NCI; GM54709; GM; NIGMS

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

**Role of the intracellular domain of the human type I interferon receptor 2 chain (*IFNAR2c*) in interferon signaling. Expression of *IFNAR2c* truncation mutants in U5A cells.**

A human cell line (U5A) lacking the type I interferon (IFN) receptor chain 2 (*IFNAR2c*) was used to determine the role of the *IFNAR2c* cytoplasmic domain in regulating IFN-dependent STAT activation, interferon-stimulated gene factor 3 (ISGF3) and c-sis-inducible factor (SIF) complex formation, gene expression, and antiproliferative effects. A panel of U5A cells expressing truncation mutants of *IFNAR2c* on their cell surface were generated for study. Janus kinase (JAK) activation was detected in all mutant cell lines; however, STAT1 and STAT2 activation was observed only in U5A cells expressing full-length *IFNAR2c* and *IFNAR2c* truncated at residue 462 (R2.462). *IFNAR2c* mutants truncated at residues 417 (R2. 417) and 346 (R2.346) or *IFNAR2c* mutant lacking tyrosine residues in its cytoplasmic domain (R2.Y-F) render the receptor inactive. A similar pattern was observed for IFN-inducible STAT activation...

... ablated in U5A, R2.Y-F, R2.417, and R2.346 cell lines. The implications are that tyrosine phosphorylation and the 462-417 region of *IFNAR2c* are independently obligatory for receptor activation. In addition, the distal 53 amino acids of the intracellular domain of *IFNAR2c* are not required for IFN-receptor mediated STAT activation, ISFG3 or SIF complex formation, induction of gene expression, and inhibition of thymidine incorporation. These data demonstrate for the first time that both tyrosine phosphorylation and a specific domain of *IFNAR2c* are required in human cells for IFN-dependent coupling of JAK activation to STAT phosphorylation, gene induction, and antiproliferative effects. In addition, human and murine cells appear to require different regions of the cytoplasmic domain of *IFNAR2c* for regulation of IFN responses.

...; Structure, Tertiary; Protein-Tyrosine Kinase--metabolism--ME; Receptors, Interferon--analysis--AN; Receptors, Interferon--genetics--GE; Signal Transduction; Trans-Activators--metabolism--ME; Transcription Factors--metabolism--ME; *Tumor* Cells, Cultured; Viral Interference

**11/3,K/6 (Item 1 from file: 5)**

DIALOG(R)File 5:Biosis Previews(R)



0014106526 BIOSIS NO.: 200300065245

**Multiple STAT activation by type I interferons is mediated by common tyrosine residues located in the cytoplasmic domain of *IFNAR2c*.**

AUTHOR: Velichko Sharlene (Reprint); Wagner T Charis (Reprint); Vogel David (Reprint); Turkson James; Jove Richard; Croze Ed (Reprint)

AUTHOR ADDRESS: Immunology, Berlex Bioscience, Richmond, CA, 94804, USA** USA

JOURNAL: Journal of Interferon and Cytokine Research 22 (Supplement 1): p S-91 2002 2002

MEDIUM: print

CONFERENCE/MEETING: Joint Meeting of the International Society for Interferon and Cytokine Research, the International Cytokine Society, the Society for Leukocyte Biology, and the European Cytokine Society on

Cytokines and Interferons Turin, Italy October 06-10, 2002; 20021006
SPONSOR: International Society for Interferon and Cytokine Research
ISSN: 1079-9907 _(ISSN print)
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

**Multiple STAT activation by type I interferons is mediated by common tyrosine residues located in the cytoplasmic domain of *IFNAR2c*.**
DESCRIPTORS:
DISEASES: *cancer*--
CHEMICALS & BIOCHEMICALS: ...*IFNAR2c*--

**11/3,K/7 (Item 2 from file: 5)**
DIALOG(R)File 5:Biosis Previews(R)


0014106470 BIOSIS NO.: 200300065189
**Enhanced expression of the interferon receptor, *IFNAR2c*, sensitizes both *cancer* cells and solid tumors to the antiproliferation effects of type I Interferons.**
AUTHOR: Wagner T Charis (Reprint); Chesney Steven K (Reprint); Velichko Sharlene (Reprint); Biroc Sandra (Reprint); Harde Dean (Reprint); Vogel David (Reprint); Croze Ed (Reprint)
AUTHOR ADDRESS: Departments of Immunology and Animal Pharmacology, Berlex Biosciences Inc., Richmond, CA, 94804, USA**USA
JOURNAL: Journal of Interferon and Cytokine Research 22 (Supplement 1): p S-73 2002 2002
MEDIUM: print
CONFERENCE/MEETING: Joint Meeting of the International Society for Interferon and Cytokine Research, the International Cytokine Society, the Society for Leukocyte Biology, and the European Cytokine Society on Cytokines and Interferons Turin, Italy October 06-10, 2002; 20021006
SPONSOR: International Society for Interferon and Cytokine Research
ISSN: 1079-9907 _(ISSN print)
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English
**Enhanced expression of the interferon receptor, *IFNAR2c*, sensitizes both *cancer* cells and solid tumors to the antiproliferation effects of type I Interferons.**
DESCRIPTORS:
...MAJOR CONCEPTS: *Tumor* Biology
...DISEASES: *cancer*--
CHEMICALS & BIOCHEMICALS: ...*IFNAR2c*--
MISCELLANEOUS TERMS: ...*tumor* prevention...

...*tumor* volume

**11/3,K/8 (Item 3 from file: 5)**
DIALOG(R)File 5:Biosis Previews(R)


0013777336 BIOSIS NO.: 200200370847
**STAT3 activation by type I interferons is mediated by specific tyrosines located in the cytoplasmic domain of the interferon receptor chain *IFNAR2c***
AUTHOR: Velichko Sharlene (Reprint); Wagner T Charis (Reprint); Vogel David (Reprint); Turkson James; Jove Richard; Croze Ed (Reprint)
AUTHOR ADDRESS: Immunology, Berlex Biosciences, 15049 San Pablo Avenue, Richmond, CA, 94804-0099, USA**USA
JOURNAL: FASEB Journal 16 (5): pA1222 March 22, 2002 2002
MEDIUM: print
CONFERENCE/MEETING: Annual Meeting of Professional Research Scientists on Experimental Biology New Orleans, Louisiana, USA April 20-24, 2002;

20020420
ISSN: 0892-6638
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

**STAT3 activation by type I interferons is mediated by specific tyrosines located in the cytoplasmic domain of the interferon receptor chain *IFNAR2c***

...ABSTRACT: roles in apoptosis. This observation places IFNs on a list of cellular modifiers that are able to regulate processes of transformation and malignant progression in *cancer*. The role of STAT1 and STAT2 in IFN signaling is well established; however, the mechanism of activation of STAT3 is unclear. The regulation of STAT3 by IFN has become increasingly important in light of recent results demonstrating oncogene-like constitutive activation of STAT3 in *cancer* cells. In this report we identify for the first time a mechanism of STAT3 activation occurring via the redundant usage of two single tyrosines present in *IFNAR2c*. STAT3 activation is measured in a human *cancer* cell line (U5A) stably expressing a number of *IFNAR2c* tyrosine mutants. IFN-dependent transcriptional factor formation (STAT3:STAT3) and STAT3 specific reporter activation are also described. In addition, it is shown that STAT3 activation...
DESCRIPTORS:
MAJOR CONCEPTS: *Tumor* Biology
...ORGANISMS: human *cancer* cell line
CHEMICALS & BIOCHEMICALS: ...*tumor* cell cytoplasmic domain tyrosines, *tumor* cell expression, type I interferon-induced STAT-3 protein activation mediator...

...*tumor* cell expression, type Interferon activation

**11/3,K/9 (Item 4 from file: 5)**
DIALOG(R)File 5:Biosis Previews(R)


0013148312 BIOSIS NO.: 200100320151
**The expression of interferon-alphareceptor 2C at diagnosis is associated with cytogenetic response in interferon-alpha-treated chronic myeloid leukemia patients**
AUTHOR: Barthe Christophe (Reprint); Mahon Francois-Xavier (Reprint); Gharbi Marie-Josee (Reprint); Fabere Carole; Bilhou-Nabera Christelle (Reprint); Hochhaus Andreas; Reiffers Josy (Reprint); Marit Gerald (Reprint)
AUTHOR ADDRESS: Hematology, University, Bordeaux, France**France
JOURNAL: Blood 96 (11 Part 1): p738a November 16, 2000 2000
MEDIUM: print
CONFERENCE/MEETING: 42nd Annual Meeting of the American Society of Hematology San Francisco, California, USA December 01-05, 2000; 20001201
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Abstract
LANGUAGE: English

...ABSTRACT: to whom an alternative therapy may be proposed. In this study, the levels of expression of both BCR-ABL and subunit 2c of IFNa receptor (*IFNaR2c*) genes were analyzed at diagnosis in 74 chronic phase CML patients treated with an IFNa monotherapy. By using blood samples, real-time quantitative PCR (LightCycler technology) was performed to quantify BCR-ABL, *IFNaR2c* and G6PDH mRNA as an external control. The results were compared with hematological and cytogenetic response to IFNa. A wide variation of BCR-ABL/G6PDH...

...range 0.18 -41.3), but no significant association with either response

to IFNa or other prognostic factors was observed. In contrast, the variation of *IFNaR2c*/G6PDH ratio at diagnosis was significantly associated with the achievement of major cytogenetic response (MCR ; < 34% Ph+ metaphases). Median values of *IFNaR2c*/G6PDH ratio for patients achieving MCR and for those who did not achieve it were 110.8% (range 9 - 612) and 64.4% (range 6...

...value), the probabilities to be in MCR at 24 months was 75 +- 19% but was 40% +-17% for the other group i.e.patients with *IFNaR2c*/G6PDH ratio < 78.8% (p = 0.024). In addition, this novel independent molecular factor combined with the achievement of complete hematological response at three months...

...90.4% +- 18% at 24 months; p = 0.00001). So, in the current study, we show for the first time that the expression level of *IFNaR2c* mRNA is variable at diagnosis in CML patients and is statistically associated with IFNa response.

DESCRIPTORS:
CHEMICALS & BIOCHEMICALS: ...diagnostic *tumor* expression, drug-induced cytogenetic *tumor* response association

?ds

| Set | Items | Description |
|---|---|---|
| S1 | 15712 | (INTERFERON (W) (THERAPY OR TREATMENT)) |
| S2 | 0 | S1 AND (IFNAR2C) |
| S3 | 665 | S1 AND REVIEW |
| S4 | 0 | S3 AND (CANCER AND RESTENOSIS) |
| S5 | 0 | S3 AND (RESTENOSIS) |
| S6 | 140 | S3 AND (CANCER) |
| S7 | 1 | S6 AND (GENE (W) THERAPY) |
| S8 | 39 | (IFNAR2C) |
| S9 | 1 | S8 AND (VECTOR) |
| S10 | 18 | S8 AND (CANCER OR TUMOR OR TUMOUR) |
| S11 | 9 | RD (unique items) |

?s s8 and (restenosis or (smooth (w) muscle))

| | | |
|---|---|---|
| | 39 | S8 |
| | 29824 | RESTENOSIS |
| | 350074 | SMOOTH |
| | 1297745 | MUSCLE |
| | 246408 | SMOOTH(W)MUSCLE |
| S12 | 0 | S8 AND (RESTENOSIS OR (SMOOTH (W) MUSCLE)) |

?s (gene (w) therapy) (s) (interferon (w) receptor?)
Processing

| | | |
|---|---|---|
| | 2403862 | GENE |
| | 5318351 | THERAPY |
| | 344736 | INTERFERON |
| | 2229494 | RECEPTOR? |
| S13 | 0 | (GENE (W) THERAPY) (S) (INTERFERON (W) RECEPTOR?) |

?logoff

30jan04 11:34:46 User259876 Session D587.2

| | | |
|---|---|---|
| | $3.89 | 1.215 DialUnits File155 |
| | $1.05 | 5 Type(s) in Format 3 |
| | $1.05 | 5 Types |
| $4.94 | | Estimated cost File155 |
| | $1.96 | 0.664 DialUnits File159 |
| $1.96 | | Estimated cost File159 |
| | $6.46 | 1.154 DialUnits File5 |
| | $8.75 | 5 Type(s) in Format 3 |
| | $8.75 | 5 Types |
| $15.21 | | Estimated cost File5 |
| | $15.74 | 1.606 DialUnits File73 |
| | $2.70 | 1 Type(s) in Format 3 |
| | $2.70 | 1 Types |
| $18.44 | | Estimated cost File73 |
| | | OneSearch, 4 files, 4.639 DialUnits FileOS |
| $2.10 | | TELNET |
| $42.65 | | Estimated cost this search |

$43.01  Estimated total session cost  4.729 DialUnits

### Status: Signed Off. (9 minutes)